UNITED STATES DISTRICT COURT

FOR THE DISTRICT OF NEW HAMPSHIRE

ANTHONY CAPPELLO, Plaintiff, v. CICCHETTI, LLC d/b/a Il Panino Italian Deli., a New Jersey corporation; RESTAURANT DEPOT, LLC., a Delaware limited liability company; D'ARRIGO BROS., CO., a California corporation; ADAM BROS. FARMING, INC., a California corporation, and; JOHN DOE 1-100 (fictitious name(s) of individual(s) or entity(ies) responsible for growing, processing, distributing, or supplying the subject romaine lettuce or any other component of the adulterated food product consumed by Anthony Cappello), Defendants.	Case No.: **PLAINTIFF'S COMPLAINT FOR DAMAGES FOR:** **1st Cause of Action: Strict Liability** **2nd Cause of Action: Breach of Warranty** **3rd Cause of Action: Negligence** **4th Cause of Action: Negligence *Per Se*** **(*DEMAND FOR JURY TRIAL*)**

Plaintiff Anthony Cappello, by and through his attorneys, Michael A. Delaney of McLane Middleton, Professional Association allege upon information and belief as follows:

PARTIES

1. The plaintiff, Anthony Cappello, is a resident of the State of New Hampshire with a residential address at 447 Joppa Hill Road, Bedford, New Hampshire.

2. Defendant Cicchetti, LLC d/b/a Il Panino Italian Deli and Catering restaurant (hereinafter "Il Panino") is a New Jersey Corporation with its principal place of business located at 244 Route 46 East (Calandra's Plaza) in Fairfield, New Jersey. Il Panino is a citizen of New Jersey. IlPanino was the manufacturer, supplier, producer, distributor, and/or seller of the adulterated food product that is the subject of this action.

3. Defendant Restaurant Depot, LLC (hereinafter "Restaurant Depot") is a Delaware limited liability company, with its principal place of business located at 1710 Whitestone Expressway, Whitestone, NY 11357, and registered agent located at 122 East 42nd Street, New York, NY 10168. Restaurant Depot is a citizen of Delaware and New York. At all times relevant, Restaurant Depot was a nationwide shipper, supplier, and distributor of produce, including the romaine lettuce that is the subject of this action.

4. Defendant D'Arrigo Bros., Co. (hereinafter "D'Arrigo") is a domestic for-profit corporation organized and existing under the laws of the State of California with its principal place of business located at 21777 Harris Road, Salinas, California. At all times relevant, D'Arrigo was a grower, harvester, and distributor of leafy green produce, including the romaine lettuce that is the subject of this action.

5. Defendant, Adam Bros. Farming, Inc. (hereinafter "Adam Bros."), is a domestic for-profit corporation organized and existing under the laws of the State of California, with its principal place of business located at 2101 Sinton Rd. in Santa Maria, California. Adam Bros. is a citizen of the State of California. Adam Bros. was the manufacturer, supplier, packager, distributor, and/or seller of the adulterated food product that is the subject of this action.

6. John Does 1–100 are currently unidentified growers, harvesters, processors, shippers, or manufacturers of the romaine lettuce that is the subject of this action.

JURISDICTION AND VENUE

7. This Court has jurisdiction over the subject matter of this action pursuant to 28 U.S.C. § 1332(a)(2) because the matter in controversy exceeds $75,000.00, exclusive of costs, and it is between Defendants that are citizens of the State of California and other foreign states and Plaintiff who is a citizen of New Hampshire. Personal jurisdiction exists over the Defendants based on the Defendants' continuous and systematic contacts with New Hampshire. It was foreseeable that the Defendants' activities would result in personal injury in the State of New Hampshire and all of the Plaintiff's claims arise out of the Defendant's forum-state activities.

8. Venue in the United States District Court for the Central District of New Hampshire is proper pursuant to 28 U.S.C. § 1391(a)(2) because a substantial part of the events or omissions giving rise to Plaintiff's claims and causes of action occurred in this judicial district and all personal injuries occurred in this judicial district, and because the Defendants were subject to personal jurisdiction in this judicial district at the time of the commencement of the action. Tortious injuries and tortious activities occurred in New Hampshire.

GENERAL ALLEGATIONS

The *E. coli* O157:H7 Bacteria: Sources, Characteristics, and Identification

9. *E. coli* is an archetypal commensal bacterial species that lives in mammalian intestines. *E. coli* O157:H7 is one of thousands of serotypes of *Escherichia coli*.[1] The combination of letters and numbers in the name *E. coli* O157:H7 refers to the specific antigens (proteins which provoke an antibody response) found on the body and tail, or flagellum,[2] respectively, and distinguish it from other types of *E. coli.*[3] Most serotypes of *E. coli* are harmless and live as normal flora in the intestines of healthy humans and animals.[4] The *E. coli* bacterium is among the most extensively studied microorganisms.[5] The testing done to distinguish *E. coli* O157:H7 from its other *E. coli* counterparts is called serotyping.[6] Pulsed-field gel electrophoresis (PFGE),[7] sometimes also referred to as genetic fingerprinting, is used to compare *E. coli* O157:H7 isolates

[1] *E. coli* bacteria were discovered in the human colon in 1885 by German bacteriologist Theodor Escherich. Feng, Peter, Stephen D. Weagant, Michael A. Grant, Enumeration of *Escherichia coli* and the Coliform Bacteria, in BACTERIOLOGICAL ANALYTICAL MANUAL (8th Ed. 2002), http://www.cfsan.fda.gov/~ebam/bam-4.html. Dr. Escherich also showed that certain strains of the bacteria were responsible for infant diarrhea and gastroenteritis, an important public health discovery. *Id.* Although the bacteria were initially called Bacterium coli, the name was later changed to *Escherichia coli* to honor its discoverer. *Id.*

[2] Not all *E. coli* are motile. For example, *E. coli* O157:H7 which lack flagella are thus *E. coli* O157:NM for non-motile.

[3] CDC, *Escherichia coli* O157:H7, General Information, Frequently Asked Questions: What is *Escherichia coli* O157:H7?, http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli_g.htm.

[4] Marion Nestle, Safe Food: Bacteria, Biotechnology, and Bioterrorism, 40-41 (1st Pub. Ed. 2004).

[5] James M. Jay, MODERN FOOD MICROBIOLOGY at 21 (6th ed. 2000). ("This is clearly the most widely studied genus of all bacteria.")

[6] Beth B. Bell, MD, MPH, *et al.* A Multistate Outbreak of *Escherichia coli* O157:H7-Associated Bloody Diarrhea and Hemolytic Uremic Syndrome from Hamburgers: The Washington Experience, 272 JAMA (No. 17) 1349, 1350 (Nov. 2, 1994) (describing the multiple step testing process used to confirm, during a 1993 outbreak, that the implicated bacteria were *E. coli* O157:H7).

[7] Jay, *supra* note 5, at 220-21 (describing in brief the PFGE testing process).

to determine if the strains are distinguishable.[8] A technique called multilocus variable-number of tandem repeat analysis (MLVA) is used to determine precise classification when it is difficult to differentiate between isolates with indistinguishable or very similar PFGE patterns.[9]

10. *E. coli* O157:H7 was first recognized as a pathogen in 1982 during an investigation into an outbreak of hemorrhagic colitis[10] associated with consumption of hamburgers from a fast food chain restaurant.[11] Retrospective examination of more than three thousand *E. coli* cultures obtained between 1973 and 1982 found only one (1) isolation with serotype O157:H7, and that was a case in 1975.[12] In the ten (10) years that followed, there were approximately thirty (30) outbreaks recorded in the United States.[13] This number is likely misleading, however, because *E. coli* O157:H7 infections did not become a reportable disease in any state until 1987 when Washington became the first state to mandate its reporting to public health authorities.[14] As a

[8] *Id.* Through PFGE testing, isolates obtained from the stool cultures of probable outbreak cases can be compared to the genetic fingerprint of the outbreak strain, confirming that the person was in fact part of the outbreak. Bell, *supra* note 6, at 1351-52. Because PFGE testing soon proved to be such a powerful outbreak investigation tool, PulseNet, a national database of PFGE test results was created. Bala Swaminathan, *et al.* PulseNet: The Molecular Subtyping Network for Foodborne Bacterial Disease Surveillance, United States, 7 Emerging Infect. Dis. (No. 3) 382, 382-89 (May-June 2001) (recounting the history of PulseNet and its effectiveness in outbreak investigation).

[9] Konno T. *et al.* Application of a multilocus variable number of tandem repeats analysis to regional outbreak surveillance of Enterohemorrhagic *Escherichia coli* O157:H7 infections. Jpn J Infect Dis. 2011 Jan; 64(1): 63-5.

[10] "[A] type of gastroenteritis in which certain strains of the bacterium *Escherichia coli* (*E. coli*) infect the large intestine and produce a toxin that causes bloody diarrhea and other serious complications." The Merck Manual of Medical Information, 2nd Home Ed. Online, http://www.merck.com/mmhe/sec09/ch122/ch122b.html.

[11] L. Riley, *et al.* Hemorrhagic Colitis Associated with a Rare *Escherichia coli* Serotype, 308 New. Eng. J. Med. 681, 684-85 (1983) (describing investigation of two outbreaks affecting at least 47 people in Oregon and Michigan both linked to apparently undercooked ground beef). Chinyu Su, MD & Lawrence J. Brandt, MD, *Escherichia coli* O157:H7 Infection in Humans, 123 Annals Intern. Med. (Issue 9), 698-707 (describing the epidemiology of the bacteria, including an account of its initial discovery).

[12] Riley, *supra* note 11 at 684. See also Patricia M. Griffin & Robert V. Tauxe, The Epidemiology of Infections Caused by *Escherichia coli* O157:H7, Other Enterohemorrhagic *E. coli*, and the Associated Hemolytic Uremic Syndrome, 13 Epidemiologic Reviews 60, 73 (1991).

[13] Peter Feng, *Escherichia coli* Serotype O157:H7: Novel Vehicles of Infection and Emergence of Phenotypic Variants, 1 Emerging Infect. Dis. (No. 2), 47, 47 (April-June 1995) (noting that, despite these earlier outbreaks, the bacteria did not receive any considerable attention until ten years later when an outbreak occurred 1993 that involved four deaths and over 700 persons infected).

[14] William E. Keene, *et al.* A Swimming-Associated Outbreak of Hemorrhagic Colitis Caused by *Escherichia coli* O157:H7 and Shigella Sonnei, 331 New Eng. J. Med. 579 (Sept. 1, 1994). *See also* Stephen M. Ostroff, MD, John M. Kobayashi, MD, MPH, and Jay H. Lewis, Infections with *Escherichia coli* O157:H7 in Washington State: The First Year of Statewide Disease Surveillance, 262 JAMA (No. 3) 355, 355 (July 21, 1989). ("It was anticipated the reporting requirement would stimulate practitioners and laboratories to screen for the organism.")

result, only the most geographically concentrated outbreak would have garnered enough notice to prompt further investigation.[15]

11. *E. coli* O157:H7's ability to induce injury in humans is a result of its ability to produce numerous virulence factors, most notably Shiga-like toxins.[16] Shiga toxin (Stx) has multiple variants (e.g., Stx1, Stx2, Stx2c), and acts like the plant toxin ricin by inhibiting protein synthesis in endothelial and other cells.[17] Shiga toxin is one of the most potent toxins known.[18] In addition to Shiga toxins, *E. coli* O157:H7 produces numerous other putative virulence factors including proteins, which aid in the attachment and colonization of the bacteria in the intestinal wall, and which can lyse red blood cells and liberate iron to help support the pathogen's metabolism.[19]

12. *E. coli* O157:H7 evolved from enteropathogenic *E. coli* serotype O55:H7, a cause of non-bloody diarrhea, through the sequential acquisition of phage-encoded Stx2, a large virulence plasmid, and additional chromosomal mutations.[20] The rate of genetic mutation of *E. coli* O157:H7 indicates that the common ancestor of current *E. coli* O157:H7 clades[21] likely existed

[15] *See* Keene, *supra* note 14 at 583. ("With cases scattered over four counties, the outbreak would probably have gone unnoticed had the cases not been routinely reported to public health agencies and investigated by them.") With improved surveillance, mandatory reporting in 48 states, and the broad recognition by public health officials that *E. coli* O157:H7 was an important and threatening pathogen, there were a total of 350 reported outbreaks from 1982-2002. Josef M. Rangel, et al. Epidemiology of *Escherichia coli* O157:H7 Outbreaks, United States, 1982-2002, 11 Emerging Infect. Dis. (No. 4) 603, 604 (April 2005).

[16] Griffin & Tauxe, *supra* note 12, at 61-62 (noting that the nomenclature came about because of the resemblance to toxins produced by Shigella dysenteries).

[17] Sanding K, Pathways followed by ricin and Shiga toxin into cells, Histochemistry and Cell Biology, vol. 117, no. 2:131-141 (2002). Endothelial cells line the interior surface of blood vessels. They are known to be extremely sensitive to *E. coli* O157:H7, which is cytotoxigenic to these cells making them a primary target during STEC infections.

[18] Johannes L, Shiga toxins—from cell biology to biomedical applications. Nat Rev Microbiol 8, 105-116 (February 2010). Suh JK, et al. Shiga Toxin Attacks Bacterial Ribosomes as Effectively as Eucaryotic Ribosomes, *Biochemistry*, 37 (26); 9394–9398 (1998).

[19] Welinder-Olsson C, Kaijser B. Enterohemorrhagic *Escherichia coli* (EHEC). Scand J. Infect Dis. 37(6-7): 405-16 (2005). *See also* USDA Food Safety Research Information Office *E. coli* O157:H7 Technical Fact Sheet: Role of 60-Megadalton Plasmid (p0157) and Potential Virulence Factors, http://fsrio.nal.usda.gov/document_fsheet.php?product_id=225.

[20] Kaper JB and Karmali MA. The Continuing Evolution of a Bacterial Pathogen. PNAS vol. 105 no. 12 4535-4536 (March 2008). Wick LM, *et al.* Evolution of genomic content in the stepwise emergence of *Escherichia coli* O157:H7. *J Bacteriol* 187:1783–1791(2005).

[21] A group of biological taxa (as species) that includes all descendants of one common ancestor.

some 20,000 years ago.[22] *E. coli* O157:H7 is a relentlessly evolving organism,[23] constantly mutating and acquiring new characteristics, including virulence factors that make the emergence of more dangerous variants a constant threat.[24] The CDC has emphasized the prospect of emerging pathogens as a significant public health threat for some time.[25]

13. Although foods of a bovine origin are the most common cause of both outbreaks and sporadic cases of *E. coli* O157:H7 infections,[26] outbreak of illnesses have been linked to a wide variety of food items. For example, produce has, since at least 1991, been the source of substantial numbers of outbreak-related *E. coli* O157:H7 infections.[27] Other unusual vehicles for *E. coli* O157:H7 outbreaks have included unpasteurized juices, yogurt, dried salami, mayonnaise, raw milk, game meats, sprouts, and raw cookie dough.[28]

14. According to a recent study, an estimated 93,094 illnesses are due to domestically acquired *E. coli* O157:H7 each year in the United States.[29] Estimates of foodborne acquired O157:H7 cases result in 2,138 hospitalizations and 20 deaths annually.[30] The colitis caused by *E. coli* O157:H7 is characterized by severe abdominal cramps, diarrhea that typically turns bloody within twenty-four (24) hours, and sometimes fevers.[31] The incubation period—which is to say

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[22] Zhang W, *et al.* Probing genomic diversity and evolution of *Escherichia coli* O157 by single nucleotide polymorphisms. *Genome Res* 16*:*757–767 (2006).
[23] Robins-Browne RM. The relentless evolution of pathogenic *Escherichia coli. Clin Infec Dis*. 41*:*793–794 (2005).
[24] Manning SD, *et al.* Variation in virulence among clades of *Escherichia coli* O157:H7 associated with disease outbreaks. PNAS vol. 105 no. 12 4868-4873 (2008). ("These results support the hypothesis that the clade 8 lineage has recently acquired novel factors that contribute to enhanced virulence. Evolutionary changes in the clade 8 subpopulation could explain its emergence in several recent foodborne outbreaks; however, it is not clear why this virulent subpopulation is increasing in prevalence.")
[25] Robert A. Tauxe, Emerging Foodborne Diseases: An Evolving Public Health Challenge, 3 Emerging Infect. Dis. (No. 4) 425, 427 (Oct.-Dec. 1997). ("After 15 years of research, we know a great deal about infections with *E. coli* O157:H7, but we still do not know how best to treat the infection, nor how the cattle (the principal source of infection for humans) themselves become infected.")
[26] CDC, Multistate Outbreak of *Escherichia coli* O157:H7 Infections Associated With Eating Ground Beef—United States, June-July 2002, 51 MMWR 637, 638 (2002) reprinted in 288 JAMA (No. 6) 690 (Aug. 14, 2002).
[27] Rangel *supra* note 15, at 605.
[28] Feng *supra* note 13, at 49. *See also* USDA Bad Bug Book, *Escherichia coli* O157:H7, http://www.fda.gov/food/foodsafety/foodborneillness/foodborneillnessfoodbornepathogensnaturaltoxins/badbugbook/ucm071284.htm.
[29] Scallan E, *et al.* Foodborne illness acquired in the United States –major pathogens, Emerging Infect. Dis. Jan. (2011), http://www.cdc.gov/EID/content/17/1/7.htm
[30] *Id.*, Table 3.
[31] Griffin & Tauxe, *supra* note 12, at 63.

the time from exposure to the onset of symptoms—in outbreaks is usually reported as three (3) to four (4) days, but may be as short as one (1) day or as long as ten (10) days.[32] Infection can occur in people of all ages but is most common in children.[33] The duration of an uncomplicated illness can range from one (1) to twelve (12) days.[34] In reported outbreaks, the rate of death is 0-2%, with rates running as high as 16-35% in outbreaks involving the elderly, like those that have occurred at nursing homes.[35]

15. What makes *E. coli* O157:H7 remarkably dangerous is its very low infectious dose[36] and how relatively difficult it is to kill these bacteria.[37] Unlike *Salmonella*, for example, which usually requires something approximating an "egregious food handling error, *E. coli* O157:H7 in ground beef that is only slightly undercooked can result in infection"[38]; as few as twenty (20) organisms may be sufficient to infect a person and, as a result, possibly kill them.[39] And unlike generic *E. coli*, the O157:H7 serotype multiplies at temperatures up to 44°F, survives freezing and thawing, is heat-resistant, grows at temperatures up to 111°F, resists drying, and can survive exposure to acidic environments.[40]

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[32] Centers for Disease Control, Division of Foodborne, Bacterial and Mycotic Diseases, *Escherichia coli* general information, http://www.cdc.gov/nczved/dfbmd/disease_listing/stec_gi.html. *See also* PROCEDURES TO INVESTIGATE FOODBORNE ILLNESS, 107 (IAFP 5th Ed. 1999) (identifying incubation period for *E. coli* O157:H7 as "1 to 10 days, typically 2 to 5").

[33] Su & Brandt *supra* note 11 ("the young are most often affected").

[34] Tauxe, *supra* note 25, at 1152.

[35] *Id.*

[36] Griffin & Tauxe, *supra* note 12, at 72. ("The general patterns of transmission in these outbreaks suggest that the infectious dose is low.")

[37] V.K. Juneja, O.P. Snyder, A.C. Williams, and B.S. Marmer, Thermal Destruction of *Escherichia coli* O157:H7 in Hamburger, 60 J. Food Prot. (vol. 10). 1163-1166 (1997) (demonstrating that, if hamburger does not get to 130°F, there is no bacterial destruction, and at 140°F, there is only a 2-log reduction of *E. coli* present).

[38] Griffin & Tauxe, *supra* note 12, at 72 (noting that, as a result, "fewer bacteria are needed to cause illness that for outbreaks of salmonellosis"). Nestle, *supra* note 4, at 41. ("Foods containing *E. coli* O17:H7 must be at temperatures high enough to kill all of them.") (italics in original)

[39] Patricia M. Griffin, *et al.* Large Outbreak of *Escherichia coli* O157:H7 Infections in the Western United States: The Big Picture, in RECENT ADVANCES IN VEROCYTOTOXIN-PRODUCING *ESCHERICHIA COLI* INFECTIONS, at 7 (M.A. Karmali & A. G. Goglio eds. 1994). ("The most probable number of *E. coli* O157:H7 was less than 20 organisms per gram.") There is some inconsistency with regard to the reported infectious dose. Compare Chryssa V. Deliganis, Death by Apple Juice: The Problem of Foodborne Illness, the Regulatory Response, and Further Suggestions for Reform, 53 Food Drug L.J. 681, 683 (1998) ("as few as ten") with Nestle, supra note 4, at 41 ("less than 50"). Regardless of these inconsistencies, everyone agrees that the infectious dose is, as Dr. Nestle has put it, "a miniscule number in bacterial terms." *Id.*

[40] Nestle, *supra* note 4, at 41.

16. And, finally, to make it even more of a threat, *E. coli* O157:H7 bacteria are easily transmitted by person-to-person contact.[41] There is also the serious risk of cross-contamination between raw meat and other food items intended to be eaten without cooking. Indeed, a principle and consistent criticism of the USDA's *E. coli* O157:H7 policy is the fact that it has failed to focus on the risks of cross-contamination versus those posed by so-called improper cooking.[42] With this pathogen, there is ultimately no margin of error. It is for this precise reason that the USDA has repeatedly rejected calls from the meat industry to hold consumers primarily responsible for *E. coli* O157:H7 infections caused, in part, by mistakes in food handling or cooking.[43]

Hemolytic Uremic Syndrome (HUS)

17. *E. coli* O157:H7 infections can lead to a severe, life-threatening complication called hemolytic uremic syndrome (HUS).[44] HUS accounts for the majority of the acute deaths and chronic injuries caused by the bacteria.[45] HUS occurs in 2-7% of victims, primarily children, with onset five to ten days after diarrhea begins.[46] It is the most common cause of renal failure in

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[41] Griffin & Tauxe, *supra* note 12, at 72. The apparent "ease of person-to-person transmission…is reminiscent of Shigella, an organism that can be transmitted by exposure to extremely few organisms." *Id.* As a result, outbreaks in places like daycare centers have proven relatively common. Rangel *supra* note 15, at 605-06 (finding that 80% of the 50 reported person-to-person outbreak from 1982-2002 occurred in daycare centers).

[42] *See, e.g.* National Academy of Science, *Escherichia coli* O157:H7 in Ground Beef: Review of a Draft Risk Assessment, Executive Summary, at 7 (noting that the lack of data concerning the impact of cross-contamination of *E. coli* O157:H7 during food preparation was a flaw in the Agency's risk-assessment), http://www.nap.edu/books/0309086272/html/.

[43] Kriefall v. Excel, 265 Wis.2d 476, 506, 665 N.W.2d 417, 433 (2003). ("Given the realities of what it saw as consumers' food-handling patterns, the [USDA] bored in on the only effective way to reduce or eliminate food-borne illness"—i.e., making sure that "the pathogen had not been present on the raw product in the first place.") (citing Pathogen Reduction, 61 Fed. Reg. at 38966).

[44] Griffin & Tauxe, *supra* note 12, at 65-68. *See also* Josefa M. Rangel, *et al. Epidemiology of* Escherichia coli *O157:H7 Outbreaks, United States, 1982-2002*, 11 Emerging Infect. Dis. (No. 4) 603 (April 2005) (noting that HUS is characterized by the diagnostic triad of hemolytic anemia—destruction of red blood cells, thrombocytopenia—low platelet count, and renal injury—destruction of nephrons often leading to kidney failure).

[45] Richard L. Siegler, MD, *The Hemolytic Uremic Syndrome*, 42 Ped. Nephrology, 1505 (Dec. 1995) (noting that the diagnostic triad of hemolytic anemia, thrombocytopenia, and acute renal failure was first described in 1955). ("[HUS] is now recognized as the most frequent cause of acute renal failure in infants and young children.") *See also* Beth P. Bell, MD, MPH, *et al. Predictors of Hemolytic Uremic Syndrome in Children During a Large Outbreak of Escherichia coli O157:H7 Infections*, 100 Pediatrics 1, 1 (July 1, 1997), at http://www.pediatrics.org/cgi/content/full/100/1/e12.

[46] Tauxe, *supra* note 25, at 1152. *See also* Nasia Safdar, MD, *et al. Risk of Hemolytic Uremic Syndrome After Treatment of* Escherichia coli *O157:H7 Enteritis: A Meta-analysis*, 288 JAMA (No. 8) 996, 996 (Aug. 28, 2002). ("*E. coli* serotype O157:H7 infection has been recognized as the most common cause of HUS in the United States, with 6% of patients developing HUS within 2 to 14 days of onset of diarrhea."). Amit X. Garg, MD, MA, *et al. Long-term Renal Prognosis of Diarrhea-Associated Hemolytic Uremic Syndrome: A Systematic Review, Meta-Analysis, and Meta-regression*, 290 JAMA (No. 10) 1360, 1360 (Sept. 10, 2003). ("Ninety percent of childhood cases of HUS are…due to Shiga-toxin producing *Escherichia coli*.")

children.[47] Approximately half of the children who suffer from HUS require dialysis, and at least 5% of those who survive have long term renal impairment.[48] The same number suffers severe brain damage.[49] While somewhat rare, serious injury to the pancreas, resulting in death or the development of diabetes, can also occur.[50] There is no cure or effective treatment for HUS.[51]

18. HUS is believed to develop when the toxin from the bacteria, known as Shiga-like toxin (SLT), enters the circulation through the inflamed bowel wall.[52] SLT, and most likely other chemical mediators, attach to receptors on the inside surface of blood vessel cells (endothelial cells) and initiate a chemical cascade that results in the formation of tiny thrombi (blood clots) within these vessels.[53] Some organs seem more susceptible, perhaps due to the presence of increased numbers of receptors, and include the kidney, pancreas, and brain.[54] By definition, when fully expressed, HUS presents with the triad of hemolytic anemia (destruction of red blood cells), thrombocytopenia (low platelet count), and renal failure (loss of kidney function).[55]

19. As already noted, there is no known therapy to halt the progression of HUS. HUS is a frightening complication that even in the best American centers has a notable mortality rate.[56] Among survivors, at least five percent will suffer end stage renal disease (ESRD) with the resultant need for dialysis or transplantation.[57] But, "[b]ecause renal failure can progress slowly over decades, the eventual incidence of ESRD cannot yet be determined."[58] Other long-term problems

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[47] Su & Brandt *supra* note 11.
[48] Safdar, *supra* note 46, at 996 (going on to conclude that administration of antibiotics to children with *E. coli* O157:H7 appeared to put them at higher risk for developing HUS).
[49] Richard L. Siegler, MD, *Postdiarrheal Shiga Toxin-Mediated Hemolytic Uremic Syndrome*, 290 JAMA (No. 10) 1379, 1379 (Sept. 10, 2003).
[50] Pierre Robitaille, *et al*., *Pancreatic Injury in the Hemolytic Uremic Syndrome*, 11 Pediatric Nephrology 631, 632 (1997) ("although mild pancreas involvement in the acute phase of HUS can be frequent").
[51] Safdar, *supra* note 46, at 996. *See also* Siegler, *supra* note 49, at 1379. ("There are no treatments of proven value, and care during the acute phase of the illness, which is merely supportive, has not changed substantially during the past 30 years.")
[52] Garg *supra* note 46, at 1360.
[53] *Id*. Siegler, *supra* note 45, at 1509-11 (describing what Dr. Siegler refers to as the "pathogenic cascade" that results in the progression from colitis to HUS).
[54] Garg *supra* note 46, at 1360. *See also* Su & Brandt, *supra* note 11, at 700.
[55] Garg, *supra* note 46, at 1360. *See also* Su & Brandt, *supra* note 11, at 700.
[56] Siegler, *supra* note 45, at 1519 (noting that in a "20-year Utah-based population study, 5% dies, and an equal number of survivors were left with end-stage renal disease (ESRD) or chronic brain damage.")
[57] Garg *supra* note 46, at 1366-67.
[58] Siegler, *supra* note 45, at 1519.

include the risk for hypertension, proteinuria (abnormal amounts of protein in the urine that can portend a decline in renal function), and reduced kidney filtration rate.[59] Since the longest available follow-up studies of HUS victims are 25 years, an accurate lifetime prognosis is not readily available and remains controversial.[60] All that can be said for certain is that HUS causes permanent injury, including loss of kidney function, and requires a lifetime of close medical monitoring.

Other Complications from *E. coli* O157:H7 Infection

20. Kidney disease from HUS is not the only complication that can be caused by *E. coli* O157:H7 infection. Long-term deficits to the gastrointestinal tract, brain, liver, heart, adrenal glands, spleen, and pancreas can also occur.

21. Specific long-term consequences can include hypertension (high blood pressure), cardiovascular disease, diabetes, central nervous system dysfunction, and reactive arthritis, among others. Chronic hypertension, related to kidney damage, occurs in 8-12% of children who survive HUS. Diabetes occurs in an estimated 3% (up to 15% in some studies) of HUS cases.

22. In some *E. coli* O157:H7 cases, colon damage is so severe, including perforation, that the colon must be removed; this can occur with or without HUS. Several other types of long-term intestinal problems can also occur, and some can require surgery years after initial illness. One specific potential complication is Irritable Bowel Syndrome, or IBS, a chronic disorder characterized by abdominal discomfort and altered bowel habits, including diarrhea and/or constipation.

23. Severe neurologic involvement during acute illness increases the likelihood of long-term central nervous system dysfunction, which can include manifestations such as seizures.

***E. coli* and Leafy Greens – A Brief History**

24. *E. coli* outbreaks associated with lettuce, specifically the "pre-washed" and "ready-to-eat" varieties, are by no means a new phenomenon. In fact, the frequency with which this country's fresh produce consuming public has been hit by outbreaks of pathogenic bacteria is

[59] *Id*. at 1519-20. *See also* Garg, *supra* note 46, at 1366-67.
[60] Garg *supra* note 46, at 1368.

astonishing. Here are just a sample of *E. coli* outbreaks based on information gathered by the Center for Science in the Public Interest, Kansas State University, and the Centers for Disease Control and Prevention:

Date	**Vehicle**	**Etiology**	**Confirmed Cases**	**States/Provinces**
July 1995	Lettuce (leafy green; red; romaine)	*E. coli* O157:H7	74	1: MT
Sept. 1995	Lettuce (romaine)	*E. coli* O157:H7	20	1: ID
Sept. 1995	Lettuce (iceberg)	*E. coli* O157:H7	30	1: ME
Oct. 1995	Lettuce (iceberg; unconfirmed)	*E. coli* O157:H7	11	1: OH
May-June 1996	Lettuce (mesclun; red leaf)	*E. coli* O157:H7	61	3: CT, IL, NY
May 1998	Salad	*E. coli* O157:H7	2	1: CA
Feb.-Mar. 1999	Lettuce (iceberg)	*E. coli* O157:H7	72	1: NE
Oct. 1999	Salad	*E. coli* O157:H7	92	3: OR, PA, OH
Oct. 2000	Lettuce	*E. coli* O157:H7	6	1: IN
Nov. 2001	Lettuce	*E. coli* O157:H7	20	1: TX
July-Aug. 2002	Lettuce (romaine)	*E. coli* O157:H7	29	2: WA, ID
Nov. 2002	Lettuce	*E. coli* O157:H7	13	1: IL
Dec. 2002	Lettuce	*E. coli* O157:H7	3	1: MN
Oct. 2003-May 2004	Lettuce (mixed salad)	*E. coli* O157:H7	57	1: CA
Apr. 2004	Spinach	*E. coli* O157:H7	16	1: CA
Nov.	Lettuce	*E. coli*	6	1: NJ

Date	Vehicle	Etiology	Confirmed Cases	States/Provinces
2004		O157:H7		
Sept. 2005	Lettuce (romaine)	*E. coli* O157:H7	32	3: MN, WI, OR
Sept. 2006	Spinach (baby)	*E. coli* O157:H7 and other serotypes	205	Multistate and Canada
Nov./Dec. 2006	Lettuce	*E. coli* O157:H7	71	4: NY, NJ, PA, DE
Nov./Dec. 2006	Lettuce	*E. coli* O157:H7	81	3: IA, MN, WI
July 2007	Lettuce	*E. coli* O157:H7	26	1: AL
May 2008	Romaine	*E. coli* O157:H7	9	1: WA
Oct. 2008	Lettuce	*E. coli* O157:H7	59	Multistate and Canada
Nov. 2008	Lettuce	*E. coli* O157:H7	130	Canada
Sept. 2009	Lettuce: Romaine or Iceberg	*E. coli* O157:H7	29	Multistate
Sept. 2009	Lettuce	*E. coli* O157:H7	10	Multistate
April 2010	Romaine	*E. coli* O145	33	5: MI, NY, OH, PA, TN
Oct. 2011	Romaine	*E. coli* O157:H7	60	Multistate
April 2012	Romaine	*E. coli* O157:H7	28	1: CA Canada
June 2012	Romaine	*E. coli* O157:H7	52	Multistate
Sept. 2012	Romaine	*E. coli* O157:H7	9	1: PA
Oct. 2012	Spinach and Spring Mix Blend	*E. coli* O157:H7	33	Multistate
Apr. 2013	Leafy Greens	*E. coli* O157:H7	14	Multistate
Aug.	Leafy Greens	*E. coli*	15	1: PA

Date	Vehicle	Etiology	Confirmed Cases	States/Provinces
2013		O157:H7		
Oct. 2013	Ready-To-Eat Salads	*E. coli* O157:H7	33	Multistate
Apr. 2014	Romaine	*E. coli* O126	4	1: MN
Apr. 2015	Leafy Greens	*E. coli* O145	7	3: MD, SC, VA
June 2016	Mesclun Mix	*E. coli* O157:H7	11	3: IL, MI, WI
Nov. 2017	Leafy Greens	*E. coli* O157:H7	67	Multistate and Canada
Mar. 2018	Romaine	*E. coli* O157:H7	240	Multistate and Canada

Another Outbreak of *E. coli* O157:H7 Linked to Romaine Lettuce

25. Illnesses started on dates ranging from October 7, 2018, to December 4, 2018. Ill people ranged in age from 1 to 84 years, with a median age of 25. Sixty-six percent of ill people were female. Of 54 people with information available, 25 (46%) were hospitalized, including two people who developed hemolytic uremic syndrome. No deaths were reported.

26. As of January 9, 2019, 62 people infected with the outbreak strain of *E. coli* O157:H7 were reported from 16 states and the District of Columbia.

PLAINTIFF'S INJURIES

27. On November 9, 2018, the Plaintiff ordered a takeout Mediterranean salad with romaine lettuce from Il Panino Italian Deli and Catering restaurant located at 244 Route 46 East (Calandra's Plaza) in Fairfield, New Jersey. Plaintiff consumed the Mediterranean salad.

28. Il Panino prepared the Mediterranean salad using romaine lettuce distributed by D'Arrigo. D'Arrigo shipped romaine products to Restaurant Depot in New Jersey. Il Panino received whole heads of romaine lettuce from Restaurant Depot and processed the lettuce as an ingredient in salads, including the salad purchased and consumed by the Plaintiff on November 9, 2018. Adam Bros. grew the romaine lettuce that Il Panino utilized to prepare the Plaintiff's' salad on November 9, 2018.

29. The salad that Plaintiff consumed on November 9, 2018, as alleged in the preceding paragraphs, was contaminated with *E. coli* O157:H7. Il Panino prepared the salad using romaine lettuce that was contaminated with *E. coli* O157:H7 at the time that it left the possession and control of Adam Bros.

30. After Plaintiff returned to his home in Bedford, New Hampshire on the weekend of November 10-11, Plaintiff was acting "off," and began experiencing some discomfort and abdominal cramps. All of the symptoms of discomfort and abdominal cramps first arose when Plaintiff was located at his home in Bedford, NH.

31. The following day, November 12, 2018, the Plaintiff had his first bloody diarrhea at approximately 2:00 AM. He developed more intense abdominal cramps and persistent diarrhea. Later in the morning on November 12, 2018, he became so sick with no improvement in symptoms that he decided to visit a local hospital, the Catholic Medical Center ("CMC"), in Manchester, New Hampshire so he could get medical attention.

32. On November 12, 2018, Plaintiff arrived at CMC, where Alan Flanagan, MD and Omar Alsamman, MD evaluated Plaintiff in the emergency department. On exam, Plaintiff appeared weak and tired and was using minimal words due to his discomfort. The main abnormal findings were abdominal softness, hypoactive bowel sounds and bloody stool. Plaintiff underwent a CT scan of his abdomen and pelvis which revealed a moderately thickened transverse colon and descending colon. His preliminary diagnosis was an infectious colitis of the transverse and descending colon. Plaintiff was admitted for observation and management. He was administered antibiotics and IV fluids

33. Plaintiff's condition continued to worsen after his admission to the hospital. Additional testing and culture of his stool revealed that Plaintiff was shiga toxin positive for *E. coli* O157:H7. He also manifested symptoms for hemolytic uremic syndrome (HUS), a condition that occurs when toxins crossed from his intestines into his bloodstream and blood vessels, compromising his red blood cell count and kidneys.

34. On November 15, 2018, three days after his admittance, Plaintiff's blood lactate levels showed persistent elevation and increasing toxicity. He also had new onset abdominal distension along with the lactate elevation. He was transferred to the Intensive Care Unit. Surgeon David S. Lewis, MD was summoned for assessment of possible toxic megacolon, a life-threatening complication of severe colon disease, and consideration of urgent exploratory laparotomy and colectomy. A flexible sigmoidoscopy showed evidence of hemorrhagic necrosis. Plaintiff also developed acute renal failure and thrombocytopenia, secondary to HUS. On November 16, 2018, to address the increasing risk of his morbidity from his severe condition, Plaintiff was transferred to the operating room for a subtotal colectomy with end ileostomy. Following removal of his colon during surgery, the dissected colon was observed to be diseased and dead.

35. Following his surgery, Plaintiff remained in the Intensive Care Unit until November 25, 2020 to receive intensive medical management and intravenous nutrition. He recuperated thereafter in the general patient population until his discharge on December 1, 2020.

36. Plaintiff stayed at CMC for a cumulative 20 days. He was discharged home to begin rehabilitation with skilled nursing and physical therapy. He was unable to ambulate independently and safely for some time. He required medication management, disease management, wound care, IV therapy, and catheter care. He was unable to leave his home for some time without the assistance of a person or medical device. His recovery progressed slowly. His ileostomy procedure required him to wear stoma bags for approximately twenty months to manage digestive waste. All of the personal injuries suffered by Plaintiff before and subsequent to the subtotal colectomy and ileostomy occurred in the State of New Hampshire. All medical treatment and surgery that resulted in the Plaintiff's loss of his colon, and his recuperation from his personal injuries, occurred in the State of New Hampshire.

37. On June 17, 2020 Plaintiff had a diagnostic sigmoidoscopy, which was performed by Dr. Rocco Ricciardi at Mass General Hospital. On September 22, 2020, Plaintiff underwent a second ileostomy closure surgery at Massachusetts General Hospital in Boston to reverse his

ileostomy. Plaintiff was hospitalized for several days. It took months for the wounds from the second surgery to heal.

38. Plaintiff continues to slowly recover from his injuries, and his colectomy resulted in permanent loss of his colon and life-altering changes to his lifestyle and quality of life.

CAUSES OF ACTION

Strict Liability – Count I

39. The Plaintiffs incorporates by reference paragraphs 1 – 38 herein.

40. At all times relevant hereto, the Defendants were the manufacturers, suppliers, packagers, distributors, and/or sellers of the adulterated food product that is the subject of this action.

41. The adulterated food product that the Defendants manufactured, supplied, packaged, distributed, and/or sold was, at the time it left the Defendants' control, defective and unreasonably dangerous for its ordinary and expected use because it contained *E. coli* O157:H7, a deadly pathogen.

42. The adulterated food product that the Defendants manufactured, supplied, packaged, distributed, and/or sold was delivered to the Plaintiffs without any change in its defective condition. The adulterated food product that the Defendants manufactured, supplied, packaged, distributed, and/or sold was used in the manner expected and intended, and was consumed by Plaintiff.

43. The Defendants owed a duty of care to the Plaintiff to manufacture, supply, package, distribute, and/or sell food that was not adulterated, that was fit for human consumption, that was reasonably safe in construction, and that was free of pathogenic bacteria or other substances injurious to human health. The Defendants breached this duty.

44. The Defendants owed a duty of care to the Plaintiff to manufacture, supply, package, distribute, and/or sell food that was fit for human consumption and that was safe to consume to the extent contemplated by a reasonable consumer. The Defendants breached this duty.

45. Plaintiff suffered injuries and damages as a direct and proximate result of the defective and unreasonably dangerous condition of the adulterated food product that the Defendants manufactured, supplied, packaged, distributed, and/or sold.

Breach of Warranty – Count II

46. The Plaintiff incorporates by reference paragraphs 1 – 45 herein.

47. The Defendants are liable to the Plaintiff for breaching express and implied warranties that they made regarding the adulterated product that the Plaintiff purchased. These express and implied warranties include the implied warranties of merchantability and/or fitness for a particular use. Specifically, the Defendants expressly warranted, through their sale of food to the public and by the statements and conduct of their employees and agents, that the food they prepared and sold was fit for human consumption and not otherwise adulterated or injurious to health.

48. The contaminated food that the Defendants sold to the Plaintiff would not pass without exception in the trade and was therefore in breach of the implied warranty of merchantability.

49. The contaminated food sold to the Plaintiff was not fit for the uses and purposes intended (i.e., human consumption); this product was therefore in breach of the implied warranty of fitness for its intended use.

50. As a direct and proximate cause of the Defendants' breach of warranties, as set forth above, Plaintiff sustained injuries and damages in an amount to be determined at trial.

Negligence – Count III

51. The Plaintiff incorporates by reference paragraphs 1 – 50 herein.

52. The Defendants owed to the Plaintiff a duty to use reasonable care in the manufacture, supply, packaging, distribution, and sale of their food product, which duty would have prevented or eliminated the risk that the Defendants' food products would become contaminated with *E. coli* O157:H7 or any other dangerous pathogen. The Defendants breached this duty and were therefore negligent.

53. The Defendants had a duty to comply with all federal, state, and local statutes, laws, regulations, safety codes, and provisions pertaining to the manufacture, distribution, storage, and sale of their food product, but failed to do so, and were therefore negligent. The Plaintiff was among the class of persons designed to be protected by these statutes, laws, regulations, safety codes, and provisions pertaining to the manufacture, distribution, storage, and sale of similar food products. The Defendants breached this duty and were therefore negligent.

54. The Defendants had a duty to properly supervise, train, and monitor their respective employees, and to ensure that their respective employees complied with all applicable statutes, laws, regulations, safety codes, and provisions pertaining to the manufacture, distribution, storage, and sale of similar food products. The Defendants breached this duty and were therefore negligent.

55. The Defendants had a duty to use ingredients, supplies, and other constituent materials that were reasonably safe, wholesome, and free of defects; that otherwise complied with applicable federal, state, and local laws, ordinances, regulations, codes, and provisions; and that were clean, free

from adulteration, and safe for human consumption. The Defendants breached this duty and were therefore negligent.

56. As a direct and proximate result of the Defendants' negligence, Plaintiff sustained injuries and damages in an amount to be determined at trial.

Negligence *Per Se* – Count IV

57. The Plaintiff incorporates by reference paragraphs 1 – 56 herein.

58. The Defendants had a duty to comply with all applicable state and federal regulations intended to ensure the purity and safety of their food products, including the requirements of the Federal Food, Drug, and Cosmetic Act (21 U.S.C. § 301, *et seq.*).

59. The Defendants breached that duty and, as a result, were negligent *per se* in their manufacture, distribution, and sale of food adulterated with *E. coli* O157:H7, a deadly pathogen.

60. As a direct and proximate result of the negligent *per se* conduct by the Defendants, Plaintiff sustained injuries and damages in an amount to be determined at trial.

PRAYER FOR RELIEF:

WHEREFORE, the Plaintiff prays as follows:

(1) For judgment against the Defendants on Count I of this Petition in an amount that is fair and reasonable, for his costs incurred, and for any other relief to which he may be entitled;

(2) For judgment against the Defendants on Count II of this Petition in an amount that is fair and reasonable, for his costs incurred, and for any other relief to which he may be entitled;

(3) For judgment against the Defendants on Count III of this Petition in an amount that is fair and reasonable, for his costs incurred, and for any other relief to which he may be entitled;

(4) For judgment against the Defendants on Count IV of this Petition in an amount that is fair and reasonable, for his costs incurred, and for any other relief to which he may be entitled;

(5) For costs of suit herein incurred; and

(6) For such other and further relief as this Court may deem proper.

DEMAND FOR JURY TRIAL

Plaintiff hereby demands a jury trial as provided by Rule 38(a) of the Federal Rules of Civil Procedure.

Dated: April 29, 2021

ANTHONY CAPPELLO

By His Attorneys

MCLANE MIDDLETON
PROFESSIONAL ASSOCIATION

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